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EDITORIAL COMMENT

Left Bundle Branch Block Does Not Mean Left Coronary Artery Block*

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How many legs does a dog have if you call the tail a leg?
Four. Calling a tail a leg doesn't make it a leg.
—Abraham Lincoln (1)

The function of the left bundle (LB) is to signal orderly left ventricular (LV) contraction. But talk is cheap, the saying goes. Told to contract, myocardial cells of the LV work harder than the LB, contraction requiring many nutrients and much oxygen, remarkably every second of every day and often more frequently. It follows that when there is coronary obstruction (the vessel supplying much of the LV is the left anterior descending [LAD] coronary artery) and energy supply is interrupted, the first to weaken and die are the working cells of the LV, not those of the LB. A focal infarction of the high septum can damage the origin of the LB before its ramification. Left bundle branch block (LBBB) can be caused by special patterns of coronary disease such as distal LAD occlusion combined with right coronary artery (RCA) obstruction, or transient bradycardia-related LBBB due to increased parasympathetic tone with RCA occlusion. As to LAD disease per se, an obstruction sufficient to impair the easy work of all the LB ramifications (i.e., to cause LBBB) would cause intolerable impairment of contraction or death of the LV and the person it serves.

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It then follows that LBBB in the coronary heart disease patient does not imply LAD occlusion, even when there is chest pain. This has long ago been suggested by pathologic studies (2,3) in which LBBB more often is seen with isolated conduction disease, as from proximal calcification or sclerosis, or seen with infiltration, toxins, or hypertrophy with fibrosis. Electrocardiographic features described in the GUSTO (Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries) trial by Sgarbossa et al. (4) can be used to diagnose acute myocardial infarction (MI) in the setting of LBBB. The major criteria are: 1) ST-segment elevation $\geq$1 mm concordant with the QRS duration (sensitivity 73%, specificity 92%); 2) ST-segment depression $\geq$1 at leads V_1, V_2, or V_3 (sensitivity 25%, specificity 96%); and 3) discordant ST-segment elevation $\geq$5 mm (sensitivity 31%, sensitivity 92%). These cannot be used to imply LAD occlusion (infarct location was not reported in their landmark paper). However, they do have useful specificity. Finally, in the case of calcific aortic stenosis, there is both impingement of calcium upon the proximal LB as well as hypertrophy and more often there is not LAD occlusion.

In this issue of the *Journal*, Strauss et al. (5) support prior pathology literature with clinical data of practical import. They studied patients with primary defibrillator implantation whose ejection fractions were <0.35 (had there been acute MI, studied at least a month subsequently). Scar size and location were measured with magnetic resonance imaging (MRI) using late gadolinium enhancement. In their cohort of 233 patients, 45 had LBBB and 19 had right bundle branch block (RBBB), and 54% had significant coronary artery disease (CAD). The RBBB patients had large scars; those with LBBB had small scars. Those with RBBB were more likely to have CAD (79% vs. 29% for LBBB patients). Eleven of the 15 CAD patients with RBBB had LAD infarcts. In the 13 CAD patients with LBBB, 6 had LAD disease and 3 had RCA + LAD. Of the 6 with only LAD disease, only 1 had an infarct seemingly causing the LBBB; in the remaining 5, the infarcts were only 5% to 16% of the LV and not likely to be the cause of the LBBB. Aided by localizing the area of the scars, their data suggests that RBBB indicates LAD occlusion with a large scar. LBBB suggests a nonischemic cardiomyopathy (among nonischemic LBBB patients, the majority had no scar). Parenthetically, they also looked at 20 hypertrophic cardiomyopathy patients who underwent alcohol septal ablation, which caused RBBB, but never LBBB, in 75%. The reader is referred to their paper for important details because they are well described. But certainly LBBB did not suggest isolated LAD disease with occlusion.

A caveat that could lessen the impact of their analysis is that the investigators used unconventional criteria for diagnosing LBBB, albeit their avant-garde criteria may be more useful, being based on prior MRI work and predicting the usefulness of biventricular pacing for heart failure. They used >140 ms in men and >130 ms in women to diagnosis LBBB. Others with left intraventricular conduction defects had intermediate-sized scars, albeit greater than those with QRS duration <120 ms (it has been suggested that those with more conventional LBBB criteria may instead have left ventricular hypertrophy with left anterior fascicular block). However, even when using more conventional or wide-reaching criteria for LBBB, scar size was 9.3% versus the 24% for RBBB patients; $p < 0.0001$. In using the MRI data reported in this issue of the *Journal* when considering or

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planning coronary interventions, one can recognize this limitation; perhaps we all ought to adapt to the new LBBB criteria used in this study. Perhaps this means, as comedian Stephen Wright told us: "I was walking down the street wearing glasses when my prescription ran out."

How does this data help select patients for catheterization or LAD intervention? American College of Cardiology/American Heart Association guidelines recommend that patients with presumably new LBBB undergo intervention (6), citing meta-analyses. However, the studies forming the basis for this guideline did not distinguish patients with LBBB from those with RBBB; the benefit from intervention might have merely been due to relief of LAD occlusion in the RBBB patients. Indeed, Lie et al. (7,8) showed long ago that acute MI in patients with previous LBBB is more commonly due to inferior MI, emphasizing the importance of using criteria specific for acute MI in the setting of LBBB such as cited by Sgarbossa et al. (4). On the other hand, patients with previous LBBB could do very well then go on to suffer proximal LAD occlusion. Or consider the patient with old LAD obstruction who does not do well because his or her LAD is filled retrograde by collaterals from the RCA. Then the RCA becomes obstructed. The resulting catastrophic MI presents with the wrong block—LBBB. Or simply consider the patient with LBBB and critical stenoses of both the LAD and RCA. Data from the Strauss et al. paper (5) support these possibilities in a sizable minority of LBBB patients. Further, timely intervention will not guarantee a life saved but clearly some patients will be rescued by eliminating significant LAD stenosis in the face of LBBB. It is important to keep in mind that, by study design, no patient with acute MI was looked at in this population receiving defibrillators. These last issues cause us to hesitate to negate the guideline that LBBB patients in the setting of acute chest pain require catheterization and appropriate intervention. Rather, they emphasize the need to look at scar location in the setting of acute MI with BBB, despite the difficulties requiring advanced technology that will not disrupt urgent catheterization. At least, however, we would argue that instead of reflex thrombolytic therapy, referral for catheterization is warranted.

Just as Mr. Lincoln taught, calling a tail a leg does not mean it is in fact a leg, and calling a BBB the left one rather than the right does not imply that it is caused by occlusion of the LAD artery.

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REFERENCES

1. Quotations Book. Available at: http://quotationsbook.com/quote/11321/#sthash.rOJYHyYj.ZBbvrsa7.dpbs. Accessed June 2013.
2. Lev M, Unger PN, Rosen KM, Bharati S. The anatomic substrate of complete left bundle branch block. Circulation 1974;50:479–86.
3. Sugiura M, Okada R, Okawa S, Shimada H. Pathohistological studies on the conduction system in 8 cases of complete left bundle branch block. Jpn Heart J 1970;11:5–16.
4. Sgarbossa EB, Pinski SL, Barbagelata A, et al., for the GUSTO-1 Investigators. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle branch block. N Engl J Med 1996;334:481–7.
5. Strauss DG, Loring Z, Selvester RH, et al. Right, but not left, bundle branch block is associated with large anteroseptal scar. J Am Coll Cardiol 2013;62:959–67.
6. Antman EM, Anbe DT, Armstrong PW, et al. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction—executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to revise the 1999 guidelines for the management of patients with acute myocardial infarction). J Am Coll Cardiol 2004;44:671–719.
7. Lie KI, Wellens HJ, Schuilenburg RM. Bundle branch block and acute myocardial infarction. In: Wellens HJ, Lie KI, Janse MJ, editors. The Conduction System of the Heart: Structure, Function and Clinical Applications. Philadelphia, PA: Lea and Febiger, 1976:666.
8. Lie KI, Wellens HJ, Schuilenburg RM, Becker AE, Durrer D. Factors influencing prognosis of bundle branch block complicating acute anteroseptal infarction: the value of His bundle recordings. Circulation 1974;50:935–41.

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